ANALYTICAL STUDY OF THYROTOXICOSIS

M.S. GENERAL SURGERY

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CERTIFICATE

This is to certify that this dissertation entitled. **"ANALYTICAL STUDY OF THYROTOXICOSIS"** is a bonafide record of the work done by **Dr. R. Lakshmi Devi** under my supervision and guidance in the department of General surgery of Tirunelveli Medical College. Tirunelveli during the period of her post graduate study from may 2007 to march 2010. For the partial fulfillment of M.S (General surgery) degree.

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INTRODUCTION

- Thyroid gland was first documented by Italians of the renaissance period.
- The term Thyroid (Greek – Thyreoeides, shield shaped) is attributed to Thomas warton.
- Goitre is derived from the Latin word Gutter which means "Throat."
- The most notable thyroid surgeons were Emil Theodar Kocher (1841 – 1917) and Theodor Billroth (1829-1894)
- Emil Theodor Kocher of Berne is regarded by many as "Father of Thyroid Surgery".
- In 1909, Kocher received the nobel prize for Medicine in recognition for his works on physiology, pathology & surgery of thyroid gland.
- Bernard Courtois discovered iodine in the ash of burnt sea weed. The first accounts of thyroid surgery dates back to Roger frugardi in 1170.
- Thyrotoxicosis is a syndrome which is caused by excessive secretion of Thyroid hormones.
- Hyperthyroidism is hyperfunctioning of thyroid with elevated levels of thyroid hormones.

AIM OF STUDY

1. Review the data regarding prevalence of thyrotoxicosis & its association with standard variables.
2. Discuss the various clinical presentations of thyrotoxicosis.
3. Discussion of etiopathogenesis in Thyrotoxicosis
4. Discussion of various treatment Modalities
5. Evaluate a cost effective treatment for a country like ours.
6. Discuss the complication of treatment and follow up.

REVIEW OF LITERATURE

ANATOMY

The Thyroid gland occupies an important position in the middleof the visceral compartment of neck, lying astride the trachea just above the thoracic inlet. It normally weighs about 15 – 20gm.

The gland consists of two symmetrical lobes, united by an isthmus of thyroid infront of 2^{nd}, 3^{rd}, and 4^{th} tracheal rings. The extent of Thyroid gland is from Thyroid Cartilage to the 5^{th} or 6^{th} tracheal ring or from C_3 - T_1.

The lobes are approximately 4cm long, 2cm wide, 20-40mm thick, with the isthmus 2-6mm thick. Each lobe is pear shaped, consisting of a narrow upper pole and broader lower pole. The thyroid gland is covered by fascia and strap muscles and more laterally, it is tucked under the diverging anterior borders of the sternocleidomastoid muscles and adjacent to the lobes on the medial side, is the carotid sheath.

Pyramidal lobe:

It is present in 80% of individuals. Usually to the left of midline extending upwards from the isthmus along the anterior surface of the thyroid cartilage. It is a remnant of thyro-glossal duct.

The gland moves upwards on deglutition because of fascial attachment. The normal gland is impalpable though it can be palpated in thin individuals.

The gland moves upwards on deglutition because:

- Thyroid is ensheathed in the pretracheal fascia
- Pretracheal fascia is condensed posteriorly to form ligament of Berry.
- Thyroid cartilage is attached to the hyoid bone through the thyrohyoid membrane.

Fascial Coverings:

The strap muscles are ensheathed by the investing layer of deep cervical fascia. These muscles are applied to the anterior surface of the gland but separated by a loose condensation of fascia derived from the Pre tracheal fascia. This false capsule covers the gland which is enclosed by its true capsule with its rich blood supply.

The pre-tracheal fascia is attached above to the thyroid cartilage and cricoid cartilage and this suspension of thyroid from the larynx accounts for its movements on deglutition.

During surgery, the investing layer of deep cervical fascia is opened in the Midline vertically which is relatively avascular. The space between the gland and strap muscles is entered by retracting the strap musscle.

The strap muscles are supplied from the cervical roots 1,2,3 via ansa cervicalis. These enter the muscles at its lateral border and on the deeper surface and the muscles can be divided transversely to facilitate access to the gland, provided they are resutured, there does not appear to be any functional impairment.

Blood Supply

Arterial supply

Artery		**Origin**
Superior thyroid Artery	⟶	External Carotid Artery
Inferior Thyroid Artery	⟶	Thyro-cervical trunk from Ist part of subclavian artery.
Thyroidea Ima Artery (or) Innominate Artery	⟶	Arch of aorta

The Superior thyroid artery descends from its origin and runs to the superior pole and as it reaches the gland it divides into Anterior and posterior branches.

The Inferior thyroid Artery passes behind the carotid sheath and runs transversely across the space between the thyroid and carotid sheath to enter the deep surface of the gland as separate branches. The recurrent laryngeal nerve usually lies posterior to these branches or some times in between or anterior to them.

Venous drainage

A rich venous plexus forms underneath the capsule and drains corresponding as follows.

Vein		**Drain**
Superior thyroid vein	⟶	Internal Jugular vein
Middle Thyroid vein	⟶	IJV
Inferior thyroid vein	⟶	Brachio-cephalic vein

The middle thyroid vein runs a short course, passes from middle of the gland and directly enter the IJV.

From the isthmus and lower pole the inferior thyroid vein forms a plexus that lies in the pre-tracheal fascia and drains into the brachio – cephalic vein.

Lymphatic drainage:

The lymphatic drainage of the thyroid is principally to the Internal Jugular nodes. The superior pole and the medial isthmus drain to the superior group and the Inferior drain the lower gland and empty into pretracheal and Para tracheal nodes.

Nerve Supply:

Sympathetic - Middle and Inferior cervical sympathetic ganglia

Para Sympathetic - Laryngeal branch of vagus

Superior laryngeal Nerve ⟶ External

Internal

Recurrent laryngeal Nerve.

The sympathetic nerves travel along the blood vessels to the thyroid and are vasomotor in action. The thyroid gland's relation to the Recurrent Laryngeal Nerve and External laryngeal nerve is of major significance. Identification of these nerves rather than avoiding should be a standard practice for the surgeon.

The recurrent laryugeal nerve supplies the intrinsic muscles of larynx except the cricothyroid which is supplied by the Ext. laryngeal nerve cricothyroid is a tensor of vocal cord and affects the pitch of voice.

Recurrent Laryugeal Nerve:

Right:

Recurrent laryngeal nerve originates from the vagus and it crosses the Ist part sub clavian Artery. The Nerve then loops the subclavian artery and ascends slight obliquely to enter the larynx at the level of cricoid cartilage and posterior to the cricothyroid muscle.

Left:

The Nerve originates from the vagus as it crosses the aortic arch and loops posteriorly around the ligamentum arteriosus before it ascends medially in the tracheo-oesophageal groove to enter the larynx.

Nerve in Tracheo-Oesophageal groove

Right - 64%

Left - 77%

Nerve in relation to Inferior thyroid artery

Posterior to artery	- Right	-	53%
	Left	-	69%

In others, the nerve is either anterior or in between the branches. The most meticulous step during thyroid surgery is during the dissection where RLN passes through the Berry's Ligament. It is here the nerve is in close proximity to the thyroid gland, tethered down by the ligament and it is here the nerve is most commonly injured.

Non-Recurrent RLN:

- Exclusively on Right side
- 1% of Individuals
- Associated with anomaly of subclavian Artery Right side
- Nerve runs directly to the larynx after its origin from the vagus.

External Laryngeal Nerve:

The nerve runs on the lateral surface of the Inferior constrictor muscle to innervate the crico-thyroid and lies in close proximity to the superior pole vessels.

Nerve supplies the cricothyroid muscle which tensors of vocal cord which affects the pitch of the voice. In 80% of individuals the nerve can be found over the cricothyroid muscle.

Histology of thyroid gland:

Microscopically the gland is divided into lobules that contain 20-40 follicles. Follicles are spherical and average $30\,\mu m$ in size. Follicle is lined by cuboidal epithelium and contains a central core of colloid secreted from the epithelial cells.

When the gland activity is more as in the case of Hyperthyroidism, the follicular epithelium is tall columnar and the colloid is reduced.

Sparsely intermingled between the follicular cells and also within the inter follicular spaces are the parafollicular or 'C' cells which secrete calcitonin.

The 'C' cells are mostly concentrated in the superior pole, reflecting their origin as neuro ectodermal cells derived from the ultimo-branchial bodies and are a part of APUD series described by pearse.

Development of thyroid:

Embryology of thyroid:

The thyroid develops as an endodermal tubular structure from the posterior aspect of fetal tongue and grows downwards in front of the developing hyoid and larynx, bifurcating and fusing with growth elements. From 4^{th} branchial pouch. The stem of the down growth forms the Thyroglossal duct whose upper end remains as foramen caecum of the tongue, the lower end forms the pyramidal lobe. Thyroglossal duct

usually atrophies but many remain in whole or in part and produce abnormalities.

It is Joined laterally by a pair of components originating from the ultimo-branchial bodies of the 4^{th} and 5^{th} pharyngeal pouches These lateral components supply the 'C' cells of thyroid which secrete calcitonin.

The superior parathyroid develops from 4^{th} pharyngeal pouch and it is in close relationship to superior aspect of thyroid.

The Inferior parathyroids develop from 3^{rd} pharyngeal pouch and is developmentally related to thymus.

PHYSIOLOGY

Iodine Metabolism

The average daily requirement of iodine is 0.1mg. Iodine in the stomach and jejunum is converted to Iodide and is absorbed. It is actively transported to thyroid by an ATP dependent process. The Normal serum to thyroid iodine ratio is about 1:5. But can be as high as 1:500.

Thyroid hormones T3 and T4 are bound to Thyroglobulin within the colloid.

Synthesis is as follows:

- Trapping of organic iodide
- Oxidation of Iodide to iodine
- Binding of iodine with tyrosine to form iodothyronine
- Coupling of Mono-iodotyrosine (MIT) and di-iodotyrosine (DIT) to form T3, T4 and free T_3

All the above steps in thyroid synthesis is accelerated by TSH, acting through a specific membrane receptor via the cyclic AMP second messenger system.

The coupling and oxidation of iodide are catalyzed by peroxidase enzyme which is inhibited by propylthiouracil and carbimazole.

The hormones so synthesized are stored in colloid within the thyroglobulin. When the hormones are required the complex is

reabsorbed into the epithelium by endocytosis and broken down by the lysosomes. This results in the formation of T4, T3, RT3, MIT, DIT.

The MIT and DIT are denominated and iodine is reused. The T3 and T4 enter the circulation and transported bound to thyroxine binding globulin (TBG) Thyroxine binding pre albumin (TBPA) and albumin.

99.98% of thyroid hormones are bound to protein and only 0.02% is free & active physiological form, rT3 is biologically inert. The circulating level of T4 to T3 is 10:1 to 20:1 T3 is more potent, less lightly bound to protein, 3-4 times more active than T4. About 75% of T3 is produced from peripheral conversion of T4 to T3 which is inhibited by β-Blockers.

Hypothalamus – Pituitary – Thyroid Axis:

Synthesis and liberation of thyroid hormone is controlled by TSH. (Thyrotropin) TSH is secreted by basophil cells of Anterior pituitary. The TSH is in turn controlled by Thyrotropin releasing hormone (TRH) secreted by Hypothalamus.

Molecular Basis of thyroid hormone action:

The thyroid hormones are transported across the plasma membrane of the tissues by ATP dependent transport system.

Uptake by the tissue is rate limited by the amount of free hormone available at the tissue level.

At the cellular level T3 is the active hormone and its activity is mediated through T3 receptors located in the cell nucleus.

OUTLINE OF THYROTOXICOSIS

Syndrome that results when excessive levels of thyroid hormones are secreted into circulation. The term Thyrotoxicosis is retained because symptoms due to raised level of hormone is not responsible for all manifestations of the disease.

Classification

I. Based on Etilogy:

A. Primary alteration within the thyroid

- Grave's disease
- Toxic Multi nodular goitre
- Toxic Adenoma

B. CNS – Disorders – increased TSH Secretion

II. Based on Hormone Secretion

With increased thyroid hormone secretion

- Grave's disease – Primary thyrotoxicosis
- Toxic nodular goitre
- Toxic Thyroid adenoma
- Jod Basedow Hyperthyroidism

Without increased thyroid Hormone Secretion:

- Subacute thyroiditis
- Factitious Hyper thyroidism

- Functioning Metastatic Ca thyroid
- Struma ovari (ovarian teratoma containing thyroid)
- Molar pregancy – increased β – HCG

Etio – Pathogenesis:

Grave's disease – (Diffuse toxic goitre)

Originally described by caleb pary – welsch physician 1825. Named after Robert graves, Ireland 1835.

- It is the most common form of Thyrotoxicosis
- Affects young adults (20-40 yrs)
- 6 times more common in females.

Extra thyroidal Manifestations:

Ophthalmopathy, pretibial Myxoedema, vitiligo, Thyroid Acropachy.

Etiology:

It is an autoimmune disorder. The disease was originally thought to be due to LATS. (Long acting thyroid stimulating antibody) described by Adams and parves in 1956.

It is recently demonstrated that the disease is due to wide range of antibodies termed as TRAb. (Thyroid receptor Antibodies). Thyroid stimulating antibodies (or) Ig are directed at TSH receptors on – follicular cells.

TSH (or) TBIA – TSH binding inhibiting Ig or Antibodies. All these Ab are grouped as TRAb.

What initiates the Graves and Antibodies production is unclear.

Various theories:

1. Defect in the T-Suppressor cells
2. Immune response is launched to altered antigen on the follicular cell surface
3. Genetic factors – Twins have increased chance probably due to increased frequency of association with HLA B8 and DR3 in caucasians. HLA – BW 35 in Japanese.

   Family H/o autoimmune disease, Graves disease, Hashimoto's disease may be associated.

PATHOLOGY

Macroscopic Appearance

Gland is diffuse, symmetrical, smoothly enlarged with increased vascularity.

Microscopic appearance:

Cells are hyperplastic with columnar epithelium, papillary projection and mitosis and with aggregates of lymphoid tissue, colloid is reduced.

Many follicles are empty. Some contain vacuolated colloid with the characteristic scalloped pattern adjacent to thyrocytes.

Toxic Multi nodular goitre:

- Secondary Thyrotoxicosis
- Toxic nodular goitre – plummer's disease

- Long standing Multi nodular goitre becomes hyperactive over long period.
- Middle or elderly aged females
- Hyperthyroidism is relatively mild
- Not associated with extra thyroidal manifestation
- Gland is nodular and eye signs less common.

In many cases it is the inter nodular tissue that is active and nodules are inactive. In some cases the nodules are active, and here the toxicosis is due to autonomous nodules as in toxic adenoma.

Solitary Toxic nodule

Toxic Adenoma

One or more nodules trapping and organifying more iodine and secreting more hormone, thereby producing hyperthyroidism independently of TSH control.

It is autonomous – not due to TRAb TSH secretion is suppressed by high levels of circulating hormones. Normal thyroid tissue surrounding the nodule is suppressed and inactive. Adenoma is usually >3cm in diameter pathogenesis of Dermopathy and ophthalmopathy is immune mediated but not due to TRAb, less well understood.

Pathogenesis of Graves ophthalmopathy:

It is an autoimmune disorder Immunologically mediated, but not due to TRAb. Orbital muscles and fibroblasts share a common antigen with thyrocytes, TSH receptor.

Fibroblasts proliferation in orbit leading to accumulation of hydrophilic glycosaminoglycans in the retro orbital tissues which leads to increased interstitial fluid content. And there is also chronic inflammatory cells infiltration in the retro-orbital tissue. All these lead to swelling of extra ocular muscle and increased retro orbital pressure leading exophthalmos and optic nerve compression.

Clinical Manifestation:

AGE - Graves disease – 20 - 40 yrs

Toxic – Multi nodular goitre – Middle (or) elderly

Toxic Adenoma - >40 yrs.

SEX - All thyroid diseases are common in females

Clinical features are the same in Grave's or toxic Multi nodular goitre except that Graves disease is associated with more severe toxicity & with extra thyroidal manifestations. There may be family H/O other autoimmune diseases.

In Grave's the onset is sudden also remissions and exacerbations and more of neurological symptoms. In toxic Multi nodular goitre. The

onset is insidious and may be also with cardiac failure (or) atrical fibrillation and more of CVS manifestation.

There is an increased caloric turnover and Hypermetabolic state. The most significant symptoms are loss of weight despite increased appetite, preference to cold and palpitation, heat intolerance and excessive sweating.

Gastro intestinal symptoms:

Diarrhoea, Weight loss despite good appetite.

CVS:

- Pedal edema, Cardiomyopathy, CCF.
- Increased Heart rate, Blood pressure,cardiac output, and Blood volume,
- Widened pulse pressure (corrigan's pulse), chest pain, dyspnoea, palpitation.

Cardiac Rhythm abnormalities:

- Sinus tachycardia
- Multiple extrasystole
- Paroxysmal atrial tachycardia and atrial fibrillation
- Persistent atrial fibrillation not responding to digoxin.

Neurological:

- Nervousness, irritability, restlessness, excitability

- Emotional liability, psychosis
- Tremor, Hyper reflexia, illsustained clonus

Muscular system:

- Muscle weakness, proximal myopathy
- Periodic paralysis (only noted in Chinese)
- Hair – Brittle and fine leading to hair loss
- Nail – onycholysis, clubbing

Reproductive System:

Amenorrhaea, oligomenorrhoea, Infertility, Spontaneous abortion, loss of libido and Impotence.

Dermatological

- Pretibial myxoedema
- Increased Sweating, pigmentation
- Vitiligo, thyroid Acropachy
- Spider naevi, palmar erythema

Other features:

Fatigue, apathy, gynaecomastia, osteoporosis (elderly)

Neck Swelling:

1. Grave's – diffuse symmetrically enlarged goitre
2. Toxic MNG – Multinodularity, firm nodule
3. Toxic Adenoma – Single palpable nodule.

Pretibial Myxoedema:

- Incidence 3-5%
- Nearly always associated with true exophthalmos and high levels of TRAb.

Infiltrative dermopathy:

- Mucin like deposit in the skin causing skin thickening.
- Symmetrically in early stage characterized by shiny red plaque of thickened skin with coarse hair and may be cyanotic when cold.
- In severe cases skin of whole leg, foot and ankles are involved, associated with clubbing of fingers and toe (Thyroid acropachy).
- Rarely requires treatment local injection of Triamcinolone or Beta methasone ointment under occlusion dressing may be effective.

Ocular Manifestation:

Some degrees of exophthalmos is common in all the cases. True exophthalmos is proptosis of eyes caused by infiltration of retrobulbar tissues with fluid, round cells and spasm of upper eyelid. Levator palpebrae superioris is partly innervated by sympathetic and its overactivity causes lid retraction and lid lag.

American Thyroid association classification of eye signs in Graves disease.

N	-	0	- No sign or Symptoms
O	-	1	- Only sign
S	-	2	- Sign and symptom
P	-	3	- Proptosis
E	-	4	- Extra ocular muscle involvement
C	-	5	- Corneal involvement
S	-	6	- Sight loss (Optic Nerve damage)

Eye Signs:

I. **Lid lag and Lid retraction**

II. Signs of Exophthalmos:

Von – Grafe's Sign- Lid lag

Dalrympte's Sign - Lid retraction and upper sclera is visible

Joffroy's Sign - Absence of forehead wrinkling when Patient looks up.

Stellwag's Sign - Infrequent blinking staring look. Widened palpebra.

Moebius Sign - Absence of eye ball convergence due to muscle weakness.

III. Opthalmoplegia

Most common involved muscle is elevators of eye – Inferior oblique and Superior Rectus Diplopia commonly occurs at upward and outward gaze.

IV. Chemosis:

Conjunctiva is edematous, congested, edema of eyelids, conjunctival infection and chemosis are aggravated by compression of ophthalmic vein causing increased lacrimation and photophobia.

Malignant Exophthalmos:

If the above mentioned features are severe and progressive it is termed Malignant exophthalmos. Papilledema, corneal ulceration and optic nerve damage & vision loss occurs.

Examination of Exophthalmos:

Naffzieger's method-patient in sitting posture with extended neck, when observed from behind, protruded eyeball can be visualized.

Exophthalmometer (Hertel):

Measures the distance between lateral bony orbital margin to anterior surface of cornea.

Comparison:

Features	Primary	Secondary
Age	20 – 40	Middle (or) Elderly
Pre-existing goitre	Not prominent	Long standing goitre
Goitre	Diffuse, smooth	Nodular
Exophthalmos	More common	Less
Extra thyroid Manifestations	Yes	No

Neurological Manifestations	More pronounced	Less
Cardiovascular Manifestations	Less	More pronounced

Unusual causes of Hyperthyroidism

Thyrotoxicosis Factitia:

Hyperthyroidism due to excessive intake of thyroxin. It occurs only if the dose exceeds the daily requirement. Doses below the daily requirement only suppress the normal Thyroid function test.

Radio active Iodine uptake – is low

Treatment - To stop the drug and treat with carbimazole and propranolol.

Jod Basedow's disease:

(Jod German word for iodine)

Diffuse toxic goitre called Basedow's disease. Large doses of iodine given to hyperplastic endemic goitre which causes temporary Hyperthyroidism.

De Quervain's thyroiditis (Auto immune thyroiditis):

During the early stage there is Hyperthyroidism due to leakage of hormones from the gland.

Functioning Metastatic thyroid carcinoma rarely cause Hyperthyroidism.

Neonatal thyrotoxicosis:

Babies born to hyper thyroid mother have high titres of TSH and TRAb. Since both TSH and TRAb will cross the placenta. It gradually subsides in about 3 wks time and Ab titre gradually comes down.

Post – Partum thyroiditis

Causes Hyperthyroidism in early stage.

T3 thyrotoxicosis:

T4 level is normal and T3 level alone is increased and RAIU – Normal. This condition commonly occurs in endemic goitre with solitary nodule.

Investigations

Assessment of thyroid function:

TSH – N 0.15 – 4.2 μu/ml

It is done by immuno assay using monoclonal antibodies.

TSH ↓ - Hyperthyroidism

TSH ↑ - Hypothyroidism

If TSH is altered, T_3, T_4 has to be measured. Many consider that TSH is the most sensitive method to assay thyroid function.

TotalT_4 and FreeT_4:

TT_4 - N 55 – 150 n mol/L

FT_4 - N 12 – 28 p mol / L

Free T_4 is increased in early Hyperthyroidism in which the Total T_4 is normal.

Total T_3 and Free T_3

TT_3 - 1.5 - 3.5 n mol/L

FT_3 - 3 – 9 p mol/L

Free T_3 is increased in early Hyperthyroidism where Total T_3 is normal. T_4 is bound to globulin, albumin & other carrier proteins.

Pregnancy, oestrogen intake and oral contraceptive pills intake all increased carrier protein level. increased in TBG level $\rightarrow\uparrow$Total T_4 and Total T_3 level leading to false +ve results, but Free T_3 and Free T_4 remains the same. So in those persons it is ideal to measure free hormone level.

In some centres, total hormone assay has been obsolete and only free hormone assay is done. But in some places it is still being done low T_3 syndrome occurs in starvation and propranolol intake.

Antibody assay:

Anti microsomal Ab and Antithyroglobulin Ab are increased in 70% of Hyperthyroidism. TRAb is detected in 90% of Graves disease.

Thyroid scan – Isotope scan (Radio active Iodine uptake study):

- Thyroid scan is carried with either Radio active I^{123} or Tc 99^{m}.
- I^{131} is used for Radio – ablation of Thyroid, Usually after 24hrs the gland takes up 16-48% of isotope is detected.

- But when the uptake is >48% → Hyperthyroidism.
- USG (or) Isotope study – is useful in grave's Disease Routine Isotope scan is inappropriate to differentiate between benign and malignancy.
- Its principle value is in toxicity due to toxic multinodular goitre or toxic nodule. Localization of over activity of the gland will differentiate between the toxic adenoma and Toxic MNG.

Grave's - Diffuse, increase in uptake

Toxic Multi nodular goitre - Internodular tissue is overactive remaining gland is inactive.

Toxic adenoma - Nodule is overactive remaining gland is suppressed.

According to uptake:

Warm	Uptake similar to adjacent thyroid tissue
Hot	Over active nodule than surrounding thyroid
Cold	Non – functional / Hypofunctional

Sl.No.	**RAIU ↑^{ed} uptake**	**Sl.No.**	**RAIU ↓^{ed} uptake**
1.	Grave's	1.	Thyroiditis – early phase
2.	Toxic adenoma	2.	Iodine induced – Jod Base dow's Thyroiditis
3.	Toxic MNG	3.	Hashimoto's thyroiditis

USG Thyroid:

High frequency USG delineates the cyst and nodules and detects unpalpable nodules.

Any thyroid nodule has to be subjected to FNAC and cysts can be diagnosed when aspiration is attempted as part of investigating a nodule on FNAC and so USG is not absolutely necessary to diagnose cyst of thyroid. The value of USG is decreasing nowadays.

FNAC

Not usually required for toxic thyroid unless there is palpable nodule and in which carcinoma cannot be ruled out.

Done with 23 – 24 Gauze needle & 10-20ml syringe. In a single prick, multiple passes are made inside the thyroid tissue.

Some slides are alcohol (70%) dried & the some are air dried & papanicolaue stained. FNAC is highly sensitive to rule out malignancy.

X-ray neck Anteroposterior and Lateral

Will show Anteroposterior compression of trachea, lateral deviation of trachea, calcification of thyroid, shadow of thyroid gland and Retro – Sternal goitre.

CT – Scan and MRI Scan

Shows Retro sternal extension

X-ray chest – PA view

Assess cardio – pulmonary status, retro-sternal extension.

Lung metastases in malignancy

ECG, ECHO

Cardiac status, and any arrhythmias can be found out.

Indirect laryngoscopy:

Vocal cord status, pre operatively for clinical and Medico – Legal purpose.

Serum Protein – Bound Iodine(PBI):

It is cheap and can be easily assessed but lacks specificity as it measures the non-hormonal forms of iodine in the blood. In euthyroid state range is 3.5 – 8 μg/100ml.

False positive - Pregnancy, person taking iodides in various forms particularly contrast media, expectorants containing iodides and oral contraceptives.

False negative - Salicylates, androgens, hydantoin like drugs and nephrotic syndrome.

Free thyroxin index:

FT index = Serum T_4 (or) PBI x T_3 uptake percent. Normal = 3.5- 8

This correlates closely with level of FreeT_4 in serum and thus accurately reflects the thyroid status of the person.

T_3 Resin uptake:

The patient serum is incubated with Radio activeT_3. So that latter becomes fixed to any thyroid binding protein not carrying the hormone.

The amount fixed can be measured & thus the number of binding sites in the serum that are unoccupied can be measured.

- Naturally in hyperthyroidism, the number of free binding sites is low and in Hyporthyroidism this number is high. The secondary binder, where the unutilized Radio acitive T_3 become fixed can be resin or thyopac.
- The fraction of labelled T_3 taken up by the resin can be compared with that of a standard serum and this test is called as "Resin uptake ratio."
- Normal = 0.91 – 1.21 µg.
- While using thyopac method one may take 100% as the mean normal value for free binding sites. In this case less than 85% will suggest Hyperthyroidism as in this case the number of free sites will be less.
- A figure of 120% suggest Hypothyroidism as the number of free sites is high.

TSH stimulation test:

Measures the response of pituitary to I.V. administered Thyrotropin releasing hormone, the Hypothalamic stimulator of TSH, TRH given in dose of 400µgm (or) 1.73µgm/sqm.

- In euthyroid person with normal pituitary function, a prompt increase in TSH level which occurs and it peaks at 20-30mts.
- In Hypothyroidism, Basal TSH ↑ - Following TRH administration the reaction is more and TSH often reaches 100-200μ/L at 30mts.
- In thyrotoxicosis TSH response absent,when TRH is administered.

Test for Hypothalamo – Pituitary Thyroid axis:

When TRH is given, I.V in a dose of 200μg to a normal person, the level of TSH increase from basal level. From1μu/ml to a mean peak of 10μu/ml at 20 mts and returns to normal in 120 mts.

- In Hypothyroidism – there is an exaggerated rise of an already elevated TSH.
- But in Hyperthyroidism – there is response of a depressed level.

T3 Suppression test (Werner):

This is used to differentiate thyrotoxicosis from other causes of raised uptake (eg) Iodine deficiency and autonomous nodules.

Serum thyroglobulin level:

Increased in thyroditis, Grave's disease, toxic multinodular goitre

MANAGEMENT

1. Antithyroid drugs
2. Surgery
3. Radio active Iodine ablation

Drugs:

I. Drugs that inhibit hormone synthesis by blocking peroxidase enzyme

- Propylthiouracil
- Carbimazole
- Methimazole

II. Drugs that inhibit iodine trapping

- Thiocyanates
- Perchlorates
- Nitrates

III. Drugs that inhibit hormone release

- Iodides of sodium, K+ iodine

IV. β- Blockers

- Propranolol
- Nadolol

Propylthiouracil & Carbimazole

Inhibit Peroxidase → inhibit iodination and coupling

Hormone synthesis is affected, also inhibits the peripheral conversion of $T_4 \rightarrow T_3$.

Carbimazole converted to its active metabolite methimazole.

In USA propylthiouracil and Methimazole are commonly used.

In UK– only Carbimazole is used.

All these drugs have no effect on underlying disease process.

	Drug	Dose / day
1.	Propylthiouracil	100 – 300mg tds
2.	Methimazole	10 – 30 mg tds
3.	Carbimazole	5-15 mg tds
4.	Propranotol	40 – 60 mg tds

Initial response is attained in 2 weeks. After attaining response the dose is reduced to maintainance dose.

Blocking replacement regimen:

Usually after giving the initial high dose, the drug is reduced to maintainance dose. But in this regimen, Initial high dose is continued and Thyroxin is supplemented. This is called Blocking and replacement regimen.

Side effects:

a) Skin rashes, pyrexia, peripheral neuritis, vasculitis, arthritis, hepatitis.

b) Sorethroat, fever

c) Granulocytopenia – reversible, if drug is discontinued

d) Agranulocytosis – Incidence 1 in 250

e) Aplastic anemia – rare

When agranulocytosis occurs, it can treated with rh – G – Colony Stimulating factor. If side effects occurs for one drug, the therapy can be switched on to another drug as there is no crossreaction between the drugs.

Advantages:

- No surgery
- No Radiation

Disadvantages

- 50% relapse rate
- Toxicity and Drug side effects
- Teratogenicity all antithyroid drugs except propylthiouracil cross the placenta and secreted in milk.
- Longterm follow – up is necessary
- Poor patient compliance unsuitable for illiterate people.

β-Blockers

Propranolol:

- No action on gland.
- Inhibits peripheral conversion of $T_4 \rightarrow T_3$

- Inhibits sympathetic activity and results in alleviation of sympathetic mediated symptoms like tremor, excitability and nervousness.
- There is improvement of toxicity and rapid symptomatic improval. It can be used for Rapid correction to euthyroid state before surgery. Dose 40mg 8th hrly.

Nadolol:

- Long acting β- Blocker
- Single daily dose of 160mg/day

Iodides:

- Inhibit hormone release
- Lugol's Iodine (5% Iodine in 10% KI solution) reduces vascularity and also makes the gland firmer and easier to handle and less bleeding during surgery. Usually given 10 days before surgery in a dose of 5 drops 8th hrly mixed with milk or water. Alternatively Pottasium Iodide tablets can be given at 60mg tds / day.

Anti-thyroid drugs used in 3 main ways:

a) As a definitive form of treatment

b) In pre-op preparation for surgery

c) In association with Radio iodine ablation.

Drug therapy:

Unsuitable for the following

- Large diffuse gland
- Toxic Multi nodular goitre

On discontinuing the drug after attaining response it results in high rate of recurrence and definite treatment in the form of surgery or Radio iodine ablation is indicated in above mentioned condition.

Radio Iodine Ablation therapy:

- I^{131} is the most commonly used drug
- Therapeutic dose higher than the tracer dose
- First used by Hertz and Roberts

Affinity of Hyperplastic thyroid gland to iodine forms the basis for treatment RI^{131} is given as oral drink, which disintegrate within gland and emits β-rays. Penetrate the other tissue for few mm only and so the RLN, parathyroid and cartilage are not affected. The γ radiation is too small to cause to any damage.

Procedure:

500 – 750 MBq (or) 10 mci (or) 8500 CGY.

- All patients should be made euthyroid before therapy with Antithyroid drugs.

- Stop the Anti thyroid drugs, about 2-3 weeks, before therapy to allow iodine uptake of thyroid. Radio active idoine given in the form of oral drink I_{131}.
- After the single dose of therapy the response rate is 75%.
- Response is slow and occurs usually in 8-12 weeks time.
- If necessary and if the response does not occur within 12hrs, the therapy can be repeated.
- Higher the initial dose given, higher is the Hypothyroidism.
- Response occur after a period of 4-12 weeks, and during this lag period. Anti thyroid drug is given. Before ablation, an isotope scan is done to assess the size of gland.

Suitable for:

- Small or moderate sized
- Relapse after drug or surgery
- In whom surgery or drugs contraindicated
- Older Patients treated with Radio active idoine Ablation.

Contra indication

Absolute	-	Pregnancy and Lactation
Relative	-	Ophthalmopathy
	-	Isolated thyroid nodule or Toxic MNG
	-	Young age – Children and adolescence

Disadvantages:

- increased incidence of leukaemia, thyroid malignancy and other malignancies.
- Long term follow up is essential
- Repeated doses may be required.

Complication

- Exacerbation of thyrotoxicosis, arrhythmias, CCF
- Thyroid storm
- Hypothyroidism
- Hyper parathyroidism
- Worsening of eye signs
- Fetal damage in pregnancy

SURGERY

Subtotal thyroidectomy:

- Leaving behind 1/8 of normal gland
- 5 cubic cm of gland is left or 4-5gm of tissue is left
- Small portion of gland left on either side of trachea

Near total thyroidectomy:

Remove one lobe completely and leave <2gm on other side. (Hartley Dunhill procedure), so that the chance of damage of RLN is reduced in subsequent surgery if there is any recurrence.

Pre op preparation:

- Carbimazole
- Propranolol
- Lugol's iodine

Recurrence Treatment

- Large diffuse glands and young patient → surgery
- Small, moderate size → RI ablation

Subtotal Thyroidectomy:

- 10% recurrence rate
- 70% developed Hypothyroidism on 10 yrs follow up
- So current consensus is to do total or Near total thyroidectomy and put them on Thyroxin.

Toxic Multi nodular goitre:

- Anti thyroid drugs or RI therapy is less effective
- Surgery is the treatment of choice
- And in this too the original standard treatment is subtotal. But now the current trend is total thyroidectomy and Thyroxin replacement.

Toxic adenoma:

Total lobectomy – Treatment of choice

RI ablation can be given in elderly and unfit patients.

Recurrent thyrotoxicosis:

Radio active idoine ablation therapy is the treatment of choice.

Thyrotoxicosis in Children:

Radio active idoine therapy is contraindicated following surgery, as there is increased recurrence as the follicular cells are active. So treat them with drugs until late teenage and in failure of surgery can be done.

Treatment of Exophthalmos:

- Sleeping propped up
- Wearing glasses during day
- Cover the eyes during the night
- Artificial tears to protect
- Guanethidine eyedrops & steroid eyedrops
- Massive doses of steroids (Prednisolone 60mg/day)

↓

Chemosis

- Orbital decompression (or) retro orbital radiation may be required to save the eye when it is in danger.
- Retro – orbital radiation by super voltage X-rays.

Total thyroidectomy should be undertaken only in patients with severe exophthalmos, when well prepared.

Comparision study of 3 Treatment Modalities

Sl.No.	Criteria	Antithyroid drugs	Surgery	RI Ablation
1.	Mortality	Nil	<1 %	Nil
2.	Speed of control	8 – 12 wk	Immediate	-
3.	Response rate	50%	85 – 95%	75%
4.	Goitre	Remains the same (or) regress	Removed	Destroyed
5.	Hypothyroidism	Occasional but temporary	10% after 1yr 30% later 70% after 10yr	50% after 10yrs
6.	Occular disease	Improved	Improvement after total thyroidectomy	If severe exophthalmos ↓ Exacerbation rate more.
7.	In patient Treatment	Not usually necessary unless severe	Essential Before surgery	Essential
8.	Discomfort	Nil	Yes	Yes
9.	Tetany	Nil	Transient – 3% Permanent 1-2%	Less
10.	Vocal cord palsy	Nil	Present	Nil
11.	Drug reaction	Yes	Less as the drugs are discontinued before surgery	Less as the drugs are discontinued.

SUBTOTAL THYROIDECTOMY

PROCEDURE

Position :

Under General anaesthesia patient is put in supine position with neck hyperextended by placing a sand bag under shoulder

Incision :

Horizontal low - collar incision is put 2 finger breadth above the sternal notch extending from sternomastoid of one side to the other lateral borders.

Steps :

1. The skin and platysma are incised
2. Upperflap raised upto thyroid cartilage, lower flap raised upto sterno clavicular joint. Deep fascia is opened vertically in the midline.
3. Strap muscles are retracted or cut in between two artery forceps.
4. Pretracheal fascia is opened to mobilize the thyroid
5. Middle thyroid vein is ligated
6. Superior thyroid pedicle is ligated close to the gland so as to avoid injury to external laryngeal nerve
7. Inferior thyroid artery is ligated away from the gland so as to avoid injury to recurrent laryngeal nerve.

8. The gland is mobilised meticulously from the thyroid bed and leaving behind fringe of tissue over the tracheo oesophageal groove to avoid recurrent laryngeal nerve injury and preserve parathyroids.
9. After complete haemostasis, wound is closed in layers with drain insitu.

Complications :

1. Haemorrhage

May be due to slipping of ligature. It causes tachycardia, Hypotension, breathlessness and compression over the trachea which inturn cause severe stridor, respiratory obstruction. As a first aid immediate release of sutures including that of deep fascia has to be done in the ward itself and pressure over the trachea was released. Then shift the patient to Operation theatre and explore under GA and bleeding poins are identified and ligated.

2. Respiratory obstruction :

Due to Haematoma or laryngeal edema

For Haematoma – needs to be evacuated immediately

For laryngeal edema – immediate emergency endotracheal intubations is done along with administration of steroid injection – Often emergency tracheostomy may be required as a life saving procedure.

3. Recurrent laryngeal nerve palsy.

If can be transient (or) permanent. Transient is 3% common. They usually recover in 3 Wks to 3 months. Often they require treatment with steroid and speech therapy. Permanent paralysis is rare.

4. Hypoparathyroidism (0.5%)

Caused due to sapsm of arteries of parathyroid glands, occurs in 2nd to 5th day. Patient will present with weakness, positive chvostek's sign, carpopedal spasm and convulsions. Serum calcium estimation is done and then 10ml of 10% calcium gluconate is given IV 8th hrly. Later supplemented by oral calcium 500 mg 8th hrly. After 3-6weeks later patient is admitted, drug is stopped and serum calcium level is repeated.

5. Thyrotoxic crisis (Thyroid storm)

Occurs in a patient inadequately prepared for thyroidectomy.They present peroperatively or within in 12-24 hrs with severe dehydration, circulatory collapse hypotension, hyperpyrexia and often cardiac failure.

Treatment:

Inj Hydrocortisone, oral antithyroid drugs. Tepid sponging of whole body, Antipyretics, Intra venous β- Blocker injection, oral iodides, large amount of IV fluids, digitoxin cardiac monitor ventilator support.

It has got high rate of mortality with critical period of 72 hrs.

6. Injury to external laryngeal nerve causes weakness of cricothyroid muscle leading to alteration in pitch of voice

7. Hypothyroidism

8. Wound infection, Stitch granuloma

9. Keloid formation.

10. Injury to trachea and oesophagus.

MATERIALS AND METHODS

This is a prospective study of randomly selected patients who presented with hyperthyroid symptoms, of toxic goitre who were diagnosed and treated at Dept of General surgery TVMCH during the period of June 2007 to November 2009.

Each patient's symptoms and signs were entered in proforma with detailed clinical examination in relation to thyroid. All the patients were subjected to basic investigations like Complete haemogram, Blood – Urea, sugar, creatinine, chest x-ray. Thyroid profile, x-ray neck and ECG. were taken for the cases. Radio-Isotope study was not done since facilities are not available in our hospital.

Since RI ablation therapy is not available in our hospital, our patients were treated with either Antithyroid drugs or surgery.

Surgery was the modality of treatment offered to all the patients.

Out of 50 cases surgery was done for only 45 cases and for remaining 5 Cases surgery was deferred due to various reasons and these patients were treated with Antithyroid drugs only.

The 45 Patients who were planned for surgery were put on antithyroid drugs as part of pre-op preparations to euthyroid state and to prevent thyrotoxic crisis. They were monitored by daily sleeping pulse chart, periodic weight monitoring and by symptomatic improval. Surgery

was done after bringing the patient to euthyroid state pre operatively with antithyroid drugs.

Pre-Operative preparation :

Most of our patients required an average dose of carbimazole 10mg 8^{th} hrly, propranolol was also given in a dose of 40mg 12^{th} hrly. For patients with severe toxic symptoms 10days prior to surgery, Lugol's iodine 5-10 drops 8^{th} hrly was given to patients. Few Pts were given colloidal iodine instead of Lugol's iodine. Night sedation was given with diazepam or alprazolam. One patient's toxic symptoms reduced only with propylthiouracil 100mg tds.

Surgery in the form of subtotal or near total or total thyroidectomy was done.All operated specimen was sent for Histopathological Examination and analysed.

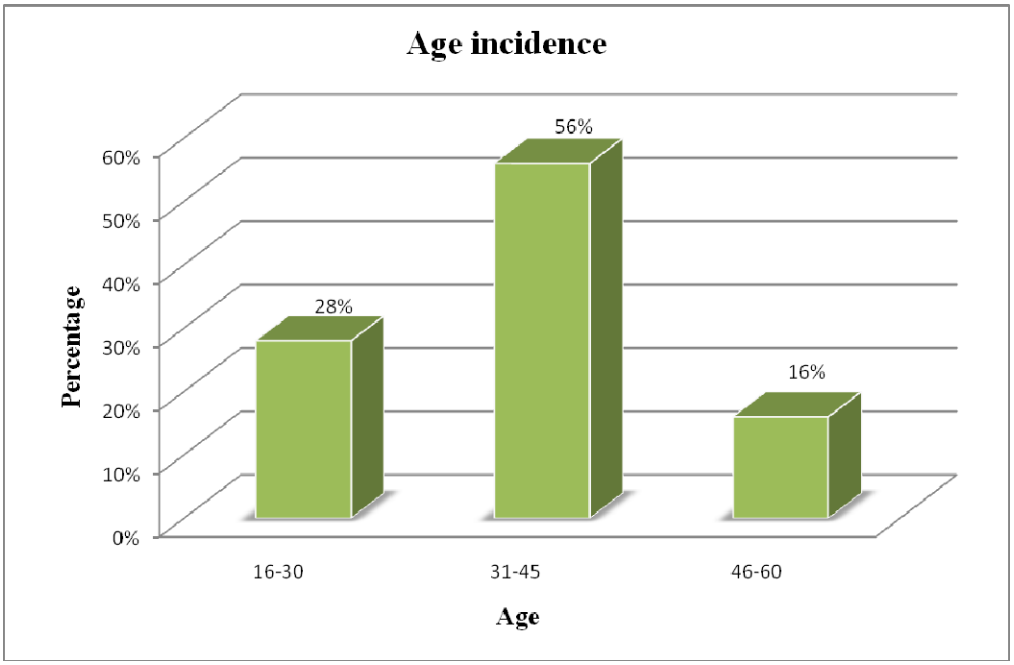
Post op complications were analysed and the patients were regularly followed up.

The Patients in whom surgery was deferred were treated with Antithyroid drugs. Total T_4 level 3 months after surgery (for 45 patients) and total T_4 level after 3 months after medical therapy (for 5 patients) were measured and analysed

All the observation were analysed and compared with results.

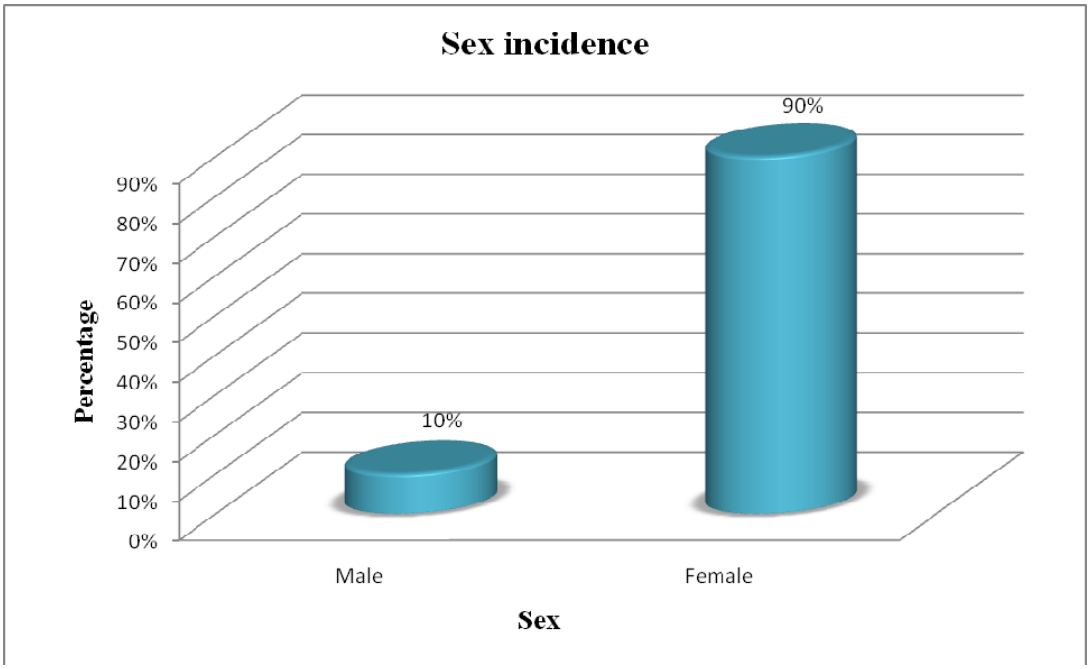
Age incidence :

S.No	Age Group	No of patients	Percentage
1	16-30	14	28%
2	31-45	28	56%
3	46-60	8	16%



Sex incidence

S.No	Sex	No of patients	Percentage
1	Male	5	10%
2	Female	45	90%

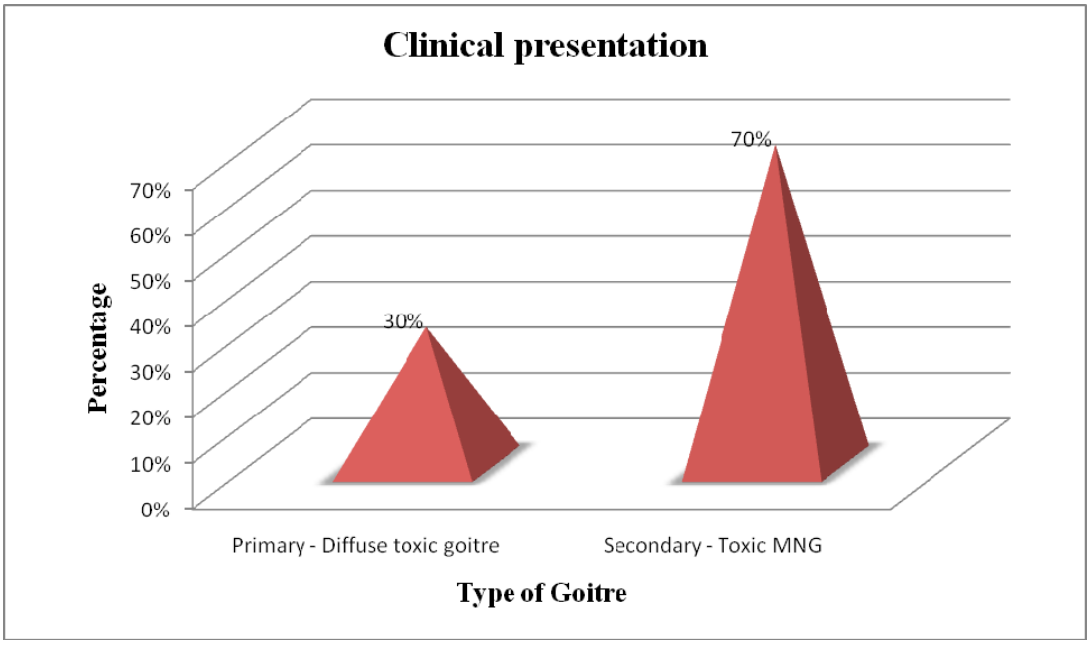


Familial Incidence

In this study no familial incidence was noted.

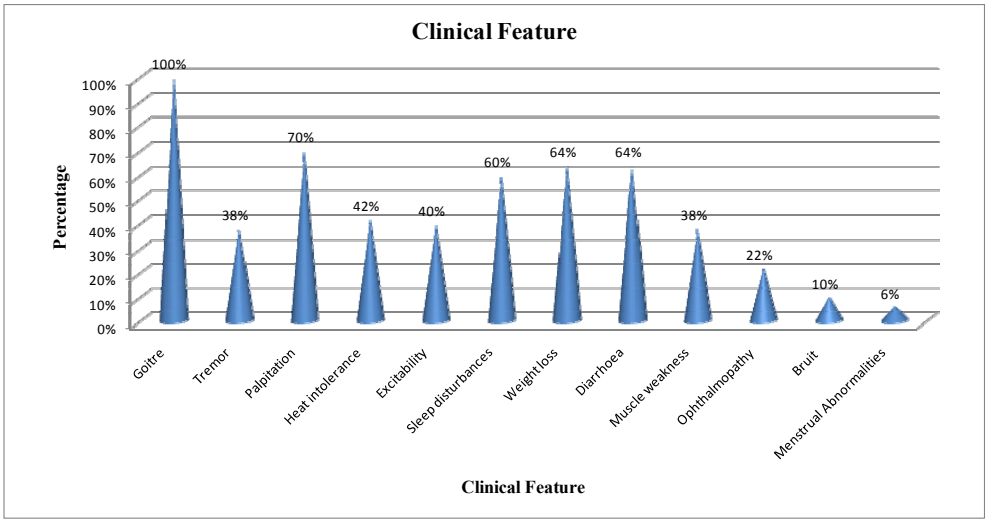
Clinical presentation :

S.No	Type of goitre	No.of Patients	Percentage
1	Primary - Diffuse toxic goitre	15	30%
2	Secondary - Toxic MNG	35	70%



Clinical feature

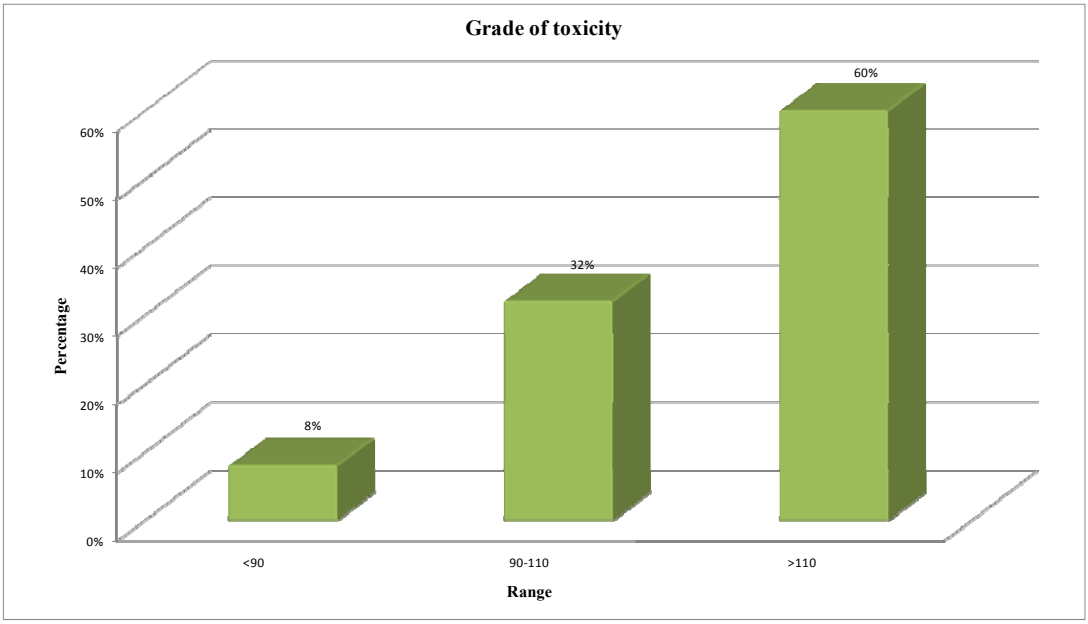
S.No	Clinical feature	No.of Patients	Percentage
1	Goitre	All	100%
2	Tremor	19	38%
3	Palpitation	35	70%
4	Heat intolerance	21	42%
5	Excitability	20	40%
6	Sleep disturbances	30	60%
7	Weight loss	32	64%
8	Diarrhoea	32	64%
9	Muscle weakness	19	38%
10	Menstrual Abnormalities	3	6%
11	Ophthalmopathy	11	22%
12	Bruit	5	10%



Few patients had menstrual abnormalities like oligomenorrhcea which got corrected once the patients were brought to Euthyroid state. None of the patients included in study had chest pain, dyspnoea, other cardiac symptoms and pressure symptoms like dysphagia, hoarseness of voice.

Grade of toxicity

S.No	Grade	Range	No.of Patients	Percentage
1	I^0	<90	4	8%
2	II^0	90-110	16	32%
3	III^o	>110	30	60%



Treatment and follow up

In our series surgery was done for 45 patients. In the rest of 5 patients surgery was deferred for the following reasons.

1 patients was not not willing for surgery

2 patients were anaesthetically unfit for surgery

2 patients were referred to Higher centre for Radio ablation therapy.

They are treated with Antithyroid drugs.

All the patients who were planned for surgery were put on antithyroid drugs as previously stated to attain euthyroid status before surgery.

Control of toxicity after pre op preparation

Duration for control of toxicity is analysed. Some patients with mild toxicity and early cases become euthyroid in 2 weeks and in other it ranged from 3-6 wks to control the toxicity. For few patients it took around 8 weeks for controlling toxicity.

Histo Pathological Examinations:

Two patients has show evidence of malignancy

- Hurthle cell carcinoma
- Papillary carcinoma thyroid

Surgical Treatment :

Surgery subtotal, near total, and Total thyroidectomy was done.

T_4 level measured in all cases 3 months after surgery.

Subtotal - 33

Near Total - 8

Total -4

Control of toxicity with drugs.

In 5 patients in whom surgery was deferred antithyroid therapy was started. They were given T. Carbimazole 10-20mg 8^{th} hrly, T.

propranolol 40mg 12th hrly. T. Diazepam for night sedation. T_4 level was measured in all the cases 3 months after therapy.

Complications

During Surgery and in post Op period the complications were encountered and are summarized,

2 patients have developed Hypoparathyroidism which manifested as

Tetany

Carpo-pedal spasm

Positive Chvostek's sign

Positive Trousseau's sign

One Patient developed symptoms on 2nd day, and other on 3rd day. They were managed as follows

Serum calcium estimation

Serum phosphorus

Treatment:

- Oral calcium
- IV calcium gluconate

Two Patients had unilateral Recurrent Laryngeal nerve palsy, It manifested as

- Hoarseness of voice
- Coughing while taking liquid drinks.

- Subsequent IDL scopy showed the paralyzed vocal cord in para median position.
- They did not develop aspiration
- They were treated with I.V. Steroids, (Dexamethasone and Hydrocortisone)
- They improved symptomatically subsequently
- They were reassured and did not develop any further complication.

One Patient developed stitch granuloma It presented as

Continuous serous discharge from a sinus in the scar for about 3 months in post operative period.

Wound was explored under local anaesthesia and there was found to be silk suture protruding in the sinus.

The sinus was cut as low as possible and the scar was excised and wound resutured.

Inference – use of non-absorbable suture material causes stitch granuloma and sinus.

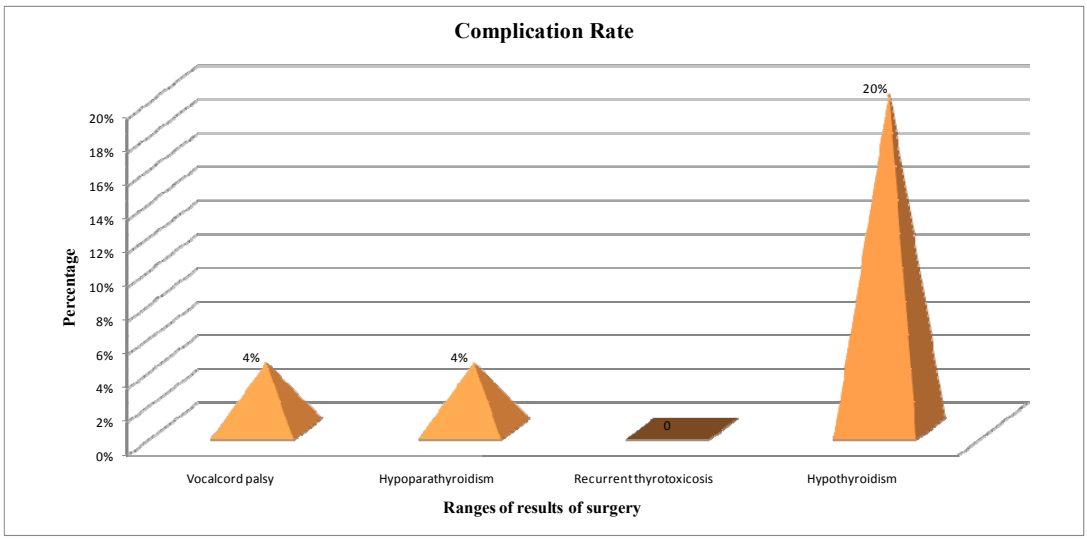
- One Patient developed subcutaneous Haematoma and was treated by aspiration.
- Two Patients developed tension Haematoma. They were immediately explored and haematoma evacuated. Bleeding sites

identified and ligated. Perfect Haemostasis obtained. Wound closed.

There was no incidence of thyroid storm or Respiratory distress or Recurrent thyrotoxicois. All the patients are living normally without any symptoms.

Complication Rate :

Sl.No	Ranges of results of surgery	From Hershmant ranges of %	In our series
1	Mortality	<3.1%	Nil
2	Vocalcord palsy	0.0 – 4.4%	4%
3	Hypoparathyroidism	0.0-3.6%	4%
4	Recurrent thyrotoxicosis	0.6-17.9%	Nil
5	Hypothyroidism	4-29.7%	20%



STATISTICAL ANALYSIS

The T_4 level is measured in all the cases before therapy and 3 months after therapy and the results were analysed.

Normal value of T_4 =55-150 nmol/L

The mean T_4 level before therapy

Before surgery in 45 cases

21.68 ± 2.42 I.S.D.

Before Antithyroid treatment in 5 cases

20.3 ±3.66 I.S.D.

The mean T_4 level after therapy

After surgery in 45 cases

4.03 ± 2.15 I.S.D.

After Antithyroid treatment

14.46 ±1.28 I.S.D.

Mean decrease of T_4 in cases treated with Antithyroid durgs is

5.84 ± 2.81 I.S.D.

The significance of difference in mean of T_4 level between patients treated medically and surgically.

P = 0.0003

This indicates the values are insignificant

DISCUSSION

- Most of the patients who were diagnosed to have thyrotoxicosis were in the age group 31-45 years. Most of the patients who presented with graves disease were in the age group of 15-30 years. Most of the cases with toxic Multi nodular goitre were in the age group of 31-45 years.
- In our study the incidence of thyrotoxicosis in females was high and the male : female ratio in our study was 1:9.
- There were no familial incidence in our study.
- Most of the patients had toxic Multi nodular goitre 35 patients and none of our cases included in study had solitary toxic nodule.
- All the patients had goitre. The most significant clinical features were palpitation, weight loss, diarrhoea, sleep disturbances, excitability and heat intolerance in the order of frequency.
- With regard to pulse rate, 60% cases had severe toxicity 3rd degree with pulse rate of >110/mt.
- TSH, Total T_4, Total T_3 level estimation was done for all cases. In all cases, the TSH was reduced, T_4 and T_3 was raised confirming the Hyperthyroid state and indicating the severity of disease. T_4 estimation repeated for all cases 3 months after therapy (both surgery and drugs).

- All the patients in whom surgery was planned were given antithyroid drugs and takenup for surgery after adequate control of toxicity. This prevented the complication of thyroid storm in the per operative and in post operative period.
- After giving Antithyroid medication, euthyroid state was attained after average of 3-4 weeks with a minimum of 2 weeks and maximum of 8 weeks.
- Most of cases underwent subtotal thyroidectomy. All the cases were followed up in post operative period until the study period.
- It was noted that none of our patients had recurrent thyrotoxicosis and there was no mortality with a little morbidity.
- The patients who underwent total thyroidectomy were given thyroxin replacement in the form of Eltroxin tablets in a dose of 0.1 mg /day.
- The Histopathology of operated specimen was studied and there was evidence of malignancy in HPE of operated specimen of two patients.
- The mean T_4 level for patients treated with surgery and Antithyroid drugs was analysed 3months after therapy. The T_4 level in the patients treated with surgery has decreased significantly to normal level (or) to just below normal level.

- But in cases treated with Antithyroid drugs the T_4 level decreased to an average level of 12-14ηu/ml which is just above the normal level.
- Out of 5 patients treated with Antithyroid drugs. Only one case came for routine follow up and other 4 patient did not turn up after 3 months. And the significance of drug therapy in them could not be assessed properly.
- From the available data in general it can be stated that surgery is the appropriate form of treatment that can be safely offered to patients with thyrotoxicosis in our setup. Since patients do not come for routine and regular follow up.

CONCLUSION

In this study, a comprehensive account of History, anatomy, physiology, with a detailed account of clinico-pathological aspects, presentations and various modalities of treatment of thyrotoxicosis have been dealt with 50 cases of thyrotoxicosis .

From the evidence it is clear that after adequate control of toxicity with anit-thyroid drugs, surgery is the modality of treatment that can be safely offered to patients with thyrotoxicosis in our hospital.

This present study confirms the observation made by the reputed authors and emphasize the importance of perfect and sufficient pre operative preparation, meticulous surgical technique, detailed knowledge about anatomy of thyroid, parathyroid and associated nerves to assure complete remission with negligible morbidity and nil mortality.

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PROFORMA

NAME : Occupation :

AGE/SEX : ADDRESS

IP.NO :

D.O.A : D.O.S D.O.D

Complaints

H/o PRESENT ILLNESS

Symptoms :

Swelling : Mode of onset

Rate of growth

Pain

↑ed appetite & Weight loss

Heat intolerance & Preference to cold

Excessive Sweating

Excitability, irritability, restlessness

Insomnia

Tremor of hands

Palpitation

Muscle weakness

Visual disturbance

Diplopia

Protrusion of eyes

Chest pain

Dyspnoea

Dysphagia, hoarseness of voice

H/O PAST ILLNESS

HT,TB,DM

PERSONAL H/O

Smoking, alcohol, intake of brassica vegetables

FAMILY H/O

MENSTRUAL H/O

OBSTETRIC H/O

TREATMENT H/O

Drug intake

Irradiation

Surgeries

G/E

Built

Nutrition

Anaemia

Dyspnoea

Jaundice

Cyanosis

Generalized lymphadenopathy

Tremor of hands & tongue

Leg edema (pretibial myxedema)

EYE SIGN – Lid retraction:

	Lid lag
Exophalmos -	von-grafe’s sign
	Joffroy’s sign
	Stellwag’s sign
	Moebius sign
	Dalrnpte’s sign
	Chemosis

Pulse rate

Sleeping pulse rate

Respiratory rate

Bp

Temperature

CVS

Rs

Abdomen

CNS

LOCAL EXAMINATION OF NECK

INSPECTION

- Swelling
- Site
- Size

- Shape
- Extent
- Borders
- Movement with deglutition
- Movement with protrusion of tongue
- Skin over the swelling
- Surface
- On sm muscle contraction
- Trachea position

PALPATION:

- Warmth
- Tenderness
- Site, size, shape
- Extent
- Borders
- Skin over swelling
- Surface
- Consistency
- Mobility
- Trachea position
- Carotid pulsation

- Kocher’s test
- Pemberton’s sign

PERCUSSION

- Over manubrium

AUSCULTATION

- Bruit
- Lymphnodes
- Skeletal system,
- Spines

DIAGNOSIS

INVESTIGATIONS

- Urine R/E
- Hb%
- Te
- Dc
- ESR
- Blood Urea
    - Sugar
    - Creatinine

ECG

ECHO:

X-ray chest PA view

X-ray neck

THYROID FUNCTION TEST

- TSH
- TT_3
- TT_4
- FT_3
- FT_4

ANTIBODY

- USG - Thyroid
- Thyroid scan
- IDL
- FNAC

TREATMENT

MEDICAL

DRUG	DOSE	DURATION	TOXICITY
1			
2			
3			

Treatment for drug toxicity

Control of toxicity attain after _____ Wks

SURGERY

Post of complication

Type – treatment

1.

2.

3.

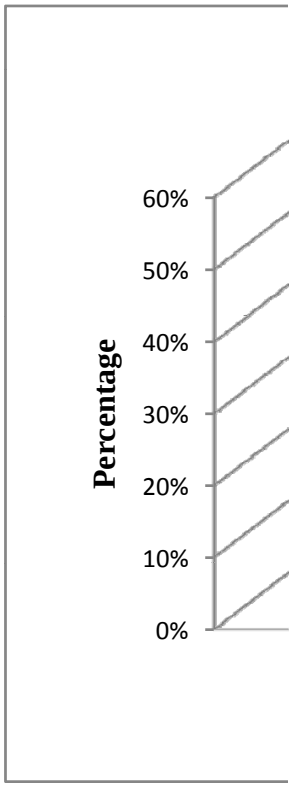
Propranolol continued in post op period for 7 days

HPE

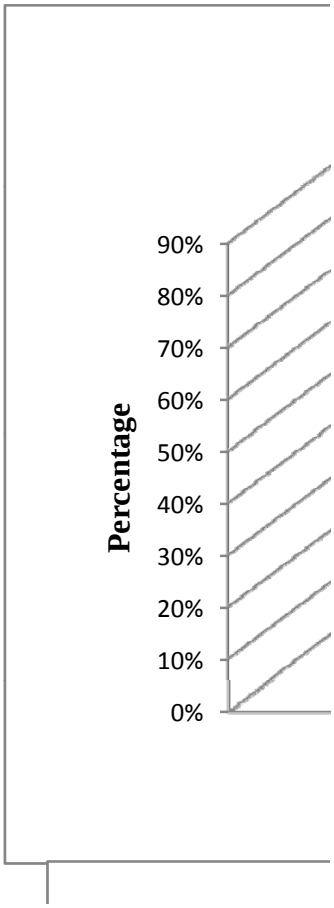
Follow -up

MASTER CHART					
S.No	**Name**	**Age/Sex**	**IP.No**	**Diagnosis**	**Treatment**
1	Velthai	27/F	12885	Diffuse toxic goitre	Sub total thyroidectomy
2	Ponkodi	20/F	22327	Diffuse toxic goitre	Sub total thyroidectomy
3	Latha	23/F	28507	Toxic MNG	Sub total thyroidectomy
4	Radha	25/F	30571	Diffuse toxic goitre	Sub total thyroidectomy
5	Kavitha	16/F	52856	Diffuse toxic goitre	Sub total thyroidectomy
6	Yasmin	16/F	48930	Diffuse toxic goitre	Total thyroidectomy
7	Chandra Vadhini	22/F	33531	Toxic MNG	Sub total thyroidectomy
8	Raja Rajeswari	26/F	10677	Diffuse toxic goitre	Sub total thyroidectomy
9	Michelammal	17/F	6908	Diffuse toxic goitre	Near total thyroidectomy
10	Petchiammal	19/F	25021	Diffuse toxic goitre	Sub total thyroidectomy
11	Vimala	27/F	29209	Toxic MNG	Antithyroid drugs
12	Maheswari	23/F	18333	Diffuse toxic goitre	Sub total thyroidectomy
13	Esakkiammal	22/F	37238	Toxic MNG	Sub total thyroidectomy
14	Vijayakumar	29/M	21239	Toxic MNG	Sub total thyroidectomy
15	Syed Ali	36/F	40325	Toxic MNG	Sub total thyroidectomy
16	Muthukumari	38/F	37056	Toxic MNG	Near total thyroidectomy
17	Avudaiammal	40/F	4958	Toxic MNG	Antithyroid drugs
18	Bharagath Nisha	35/F	15185	Diffuse toxic goitre	Total thyroidectomy
19	Lakshmi	32/F	35162	Toxic MNG	Sub total thyroidectomy
20	Kannaki	34/F	36368	Toxic MNG	Sub total thyroidectomy
21	Eswari	34/F	41725	Toxic MNG	Sub total thyroidectomy
22	Jameel	38/F	6747	Toxic MNG	Near total thyroidectomy
23	Mydeen	45/F	29394	Toxic MNG	Sub total thyroidectomy
24	Varisaimeeral	40/F	10706	Diffuse toxic goitre	Sub total thyroidectomy
25	Rani	37/F	33627	Toxic MNG	Sub total thyroidectomy
26	Thangavel	44/M	38879	Toxic MNG	Sub total thyroidectomy
27	Syed path	34/F	26380	Toxic MNG	Near total thyroidectomy
28	Saraswathi	40/F	35786	Toxic MNG	Sub total thyroidectomy
29	Guruvammal	34/F	39036	Toxic MNG	Near total thyroidectomy
30	Elaika	43/F	43309	Toxic MNG	Sub total thyroidectomy
31	Sakthivel	31/M	45623	Diffuse toxic goitre	Total thyroidectomy
32	Santhanamari	34/F	46740	Diffuse toxic goitre	Sub total thyroidectomy
33	Essakidurai	45/M	16145	Diffuse toxic goitre	Sub total thyroidectomy
34	Vasantha kumari	44/F	17329	Diffuse toxic goitre	Near total thyroidectomy
35	Ramalakshmi	33/F	29515	Diffuse toxic goitre	Sub total thyroidectomy
36	Esakkiammal	35/F	39407	Toxic MNG	Sub total thyroidectomy
37	Devi	31/F	4344	Toxic MNG	Sub total thyroidectomy
38	Maliga	34/F	31272	Toxic MNG	Antithyroid drugs
39	Manikaadan	32/M	15093	Toxic MNG	Sub total thyroidectomy
40	Chinnathai	42/F	36462	Toxic MNG	Sub total thyroidectomy
41	Pappa	40/F	2343	Diffuse toxic goitre	Sub total thyroidectomy
42	Chandra	43/F	42707	Toxic MNG	Antithyroid drugs
43	Arumugaammal	47/F	55071	Diffuse toxic goitre	Total thyroidectomy
44	Pappathi	53/F	53914	Toxic MNG	Antithyroid drugs
45	Kuthala Kannammal	35/F	2698	Toxic MNG	Near total thyroidectomy
46	Mareeswari	52/F	5689	Toxic MNG	Near total thyroidectomy
47	Parvuthi	55/F	15833	Toxic MNG	Sub total thyroidectomy
48	Sumitra	55/F	15784	Toxic MNG	Sub total thyroidectomy
49	Perumal	47/F	24163	Toxic MNG	Sub total thyroidectomy
50	Bhagavathi	50/F	44971	Toxic MNG	Sub total thyroidectomy

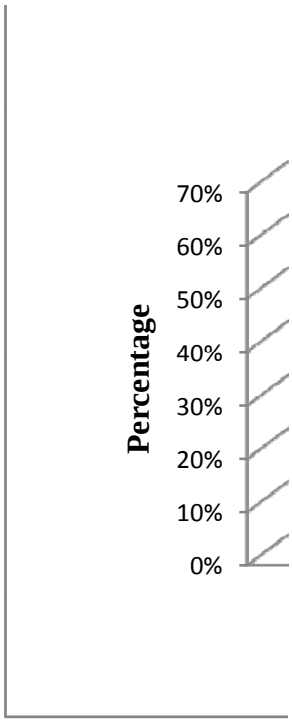
	Age Group		Percentage
	16-30		28%
	31-45		56%
	46-60		16%



	Sex		Percentage
	Male		10%
	Female		90%

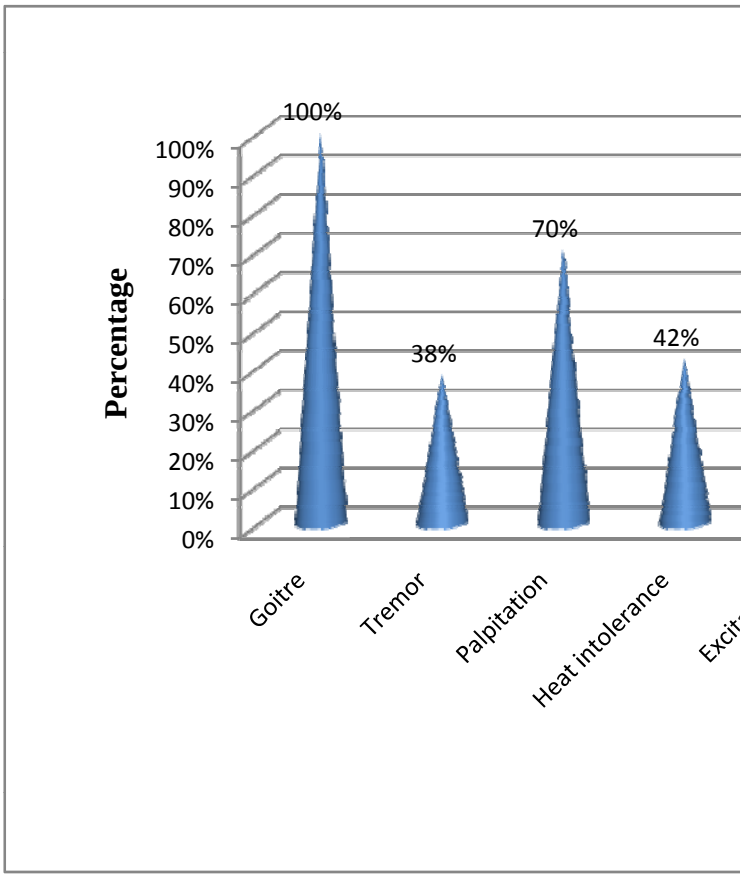


	Type of Goitre		Percentage
	Primary - Diffuse toxic goitre		30%
	Secondary - Toxic MNG		70%

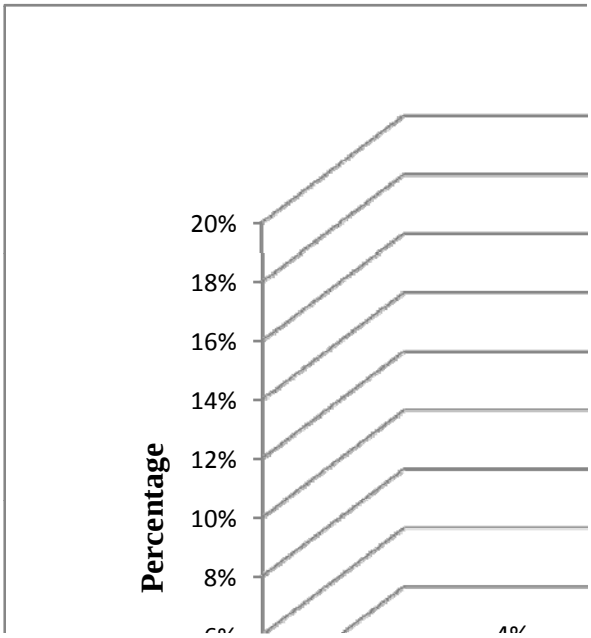


	Clinical Feature		Percentage
	Goitre		100%
	Tremor		38%
	Palpitation		70%
	Heat intolerance		42%
	Excitability		40%

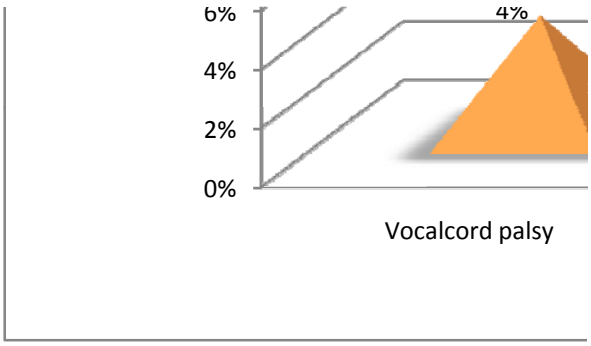
	Sleep disturbances		60%
	Weight loss		64%
	Diarrhoea		64%
	Muscle weakness		38%
	Ophthalmopathy		22%
	Bruit		10%
	Menstrual Abnormalities		6%



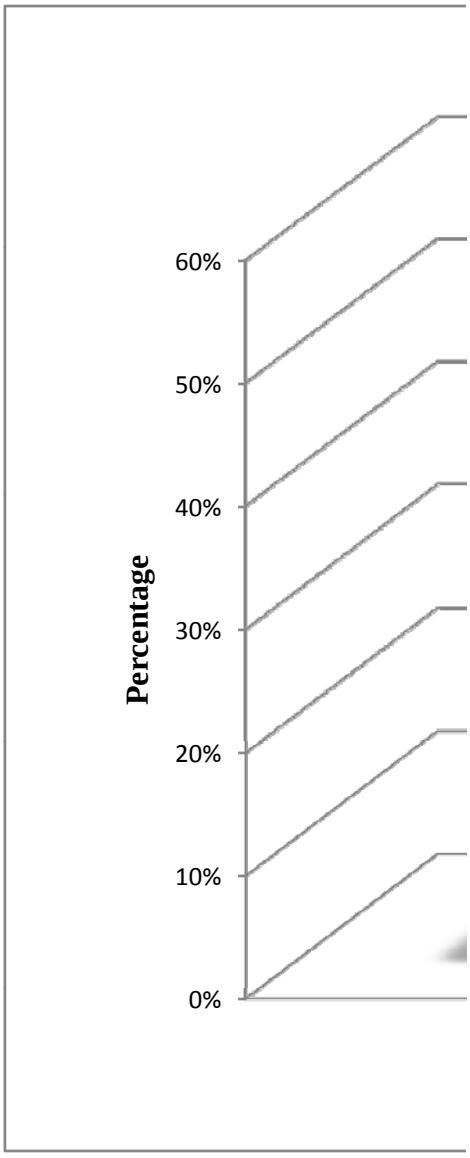
	Ranges of results of surgery		**In our series**
	Mortality		Nil
	Vocalcord palsy		4%

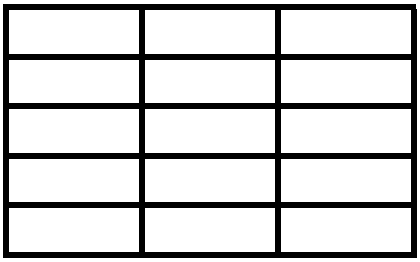


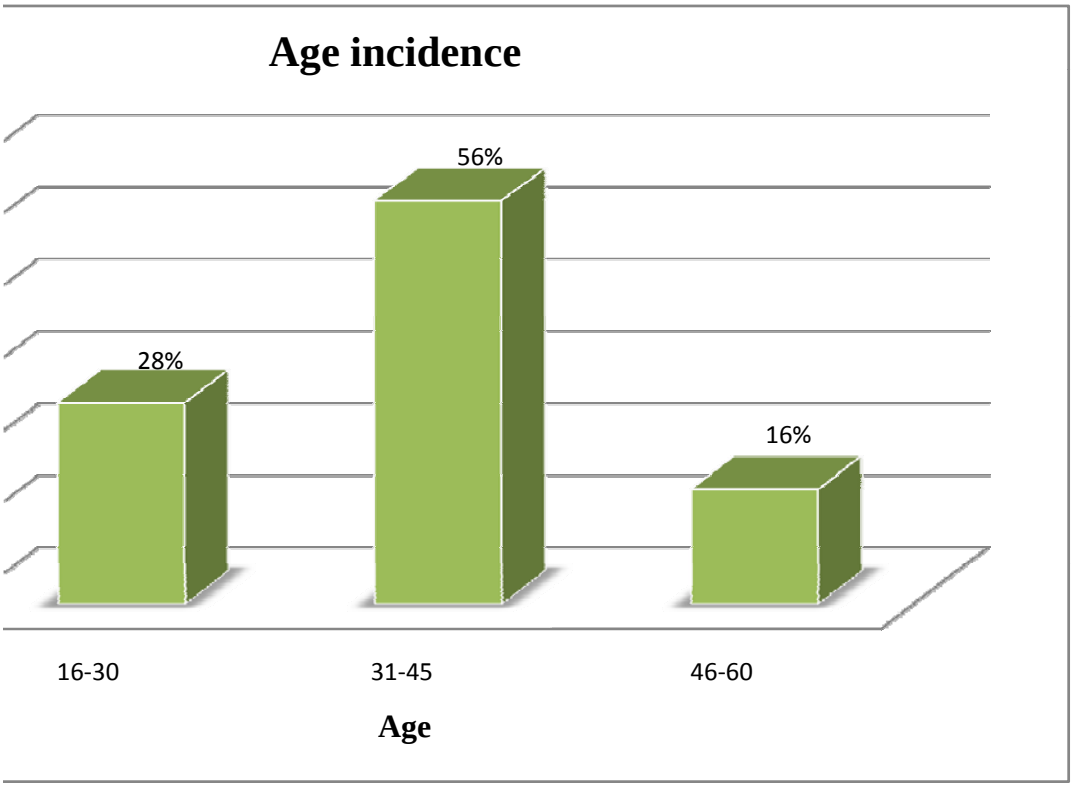
	Hypoparathyroidism		4%
	Recurrent thyrotoxicosis		Nil
	Hypothyroidism		20%

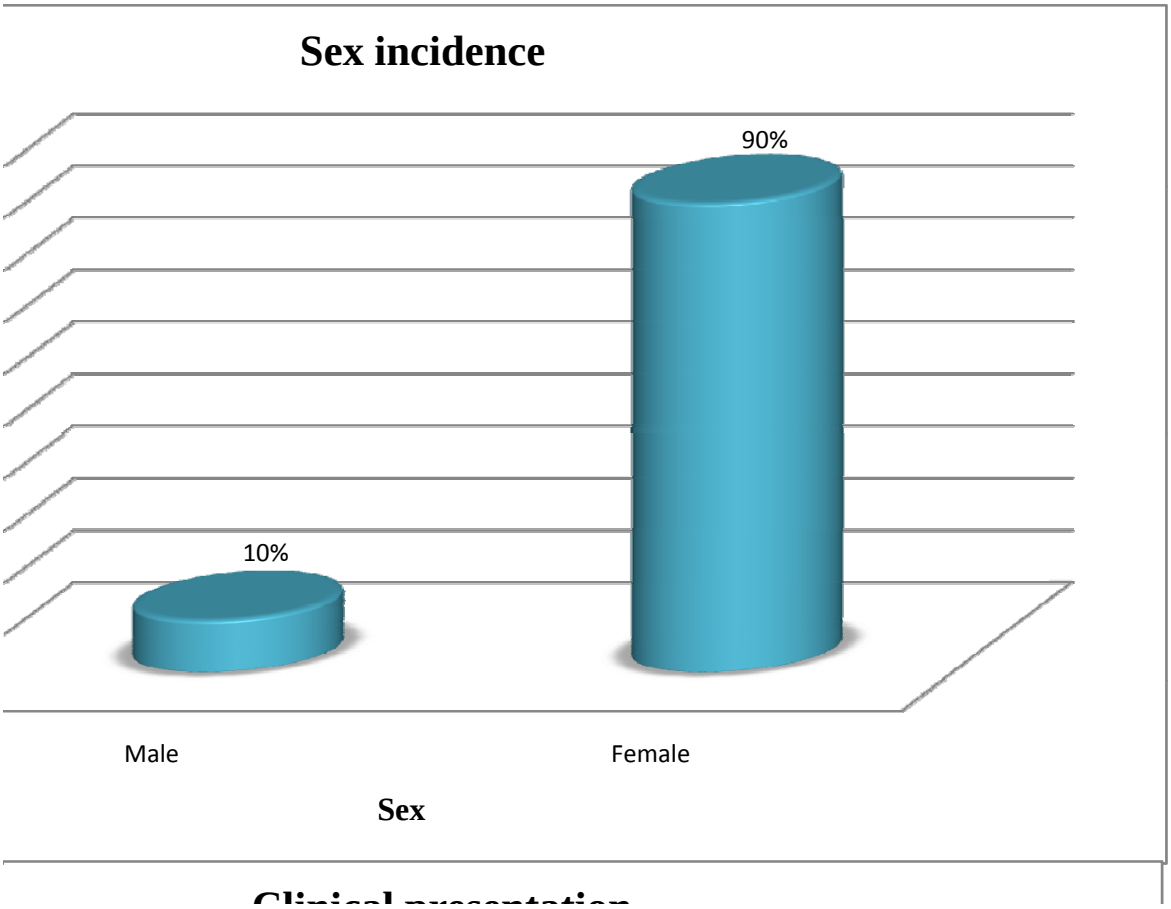


		Range		Percentage
		<90		8%
		90-110		32%
		>110		60%

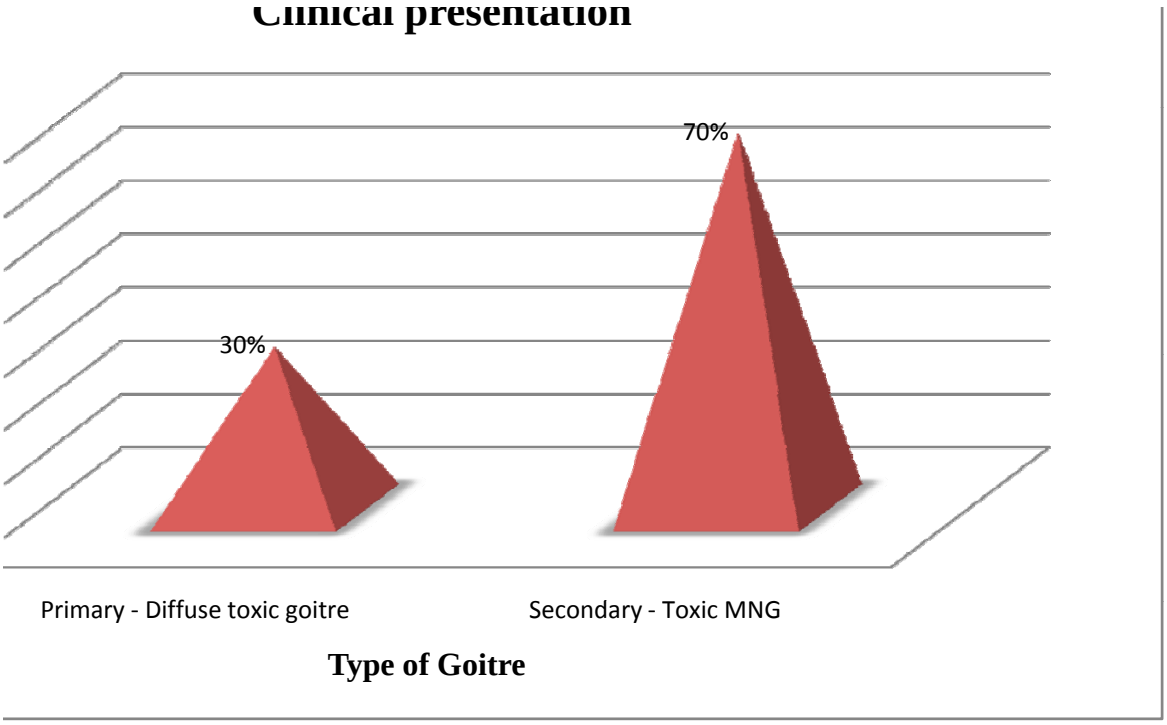


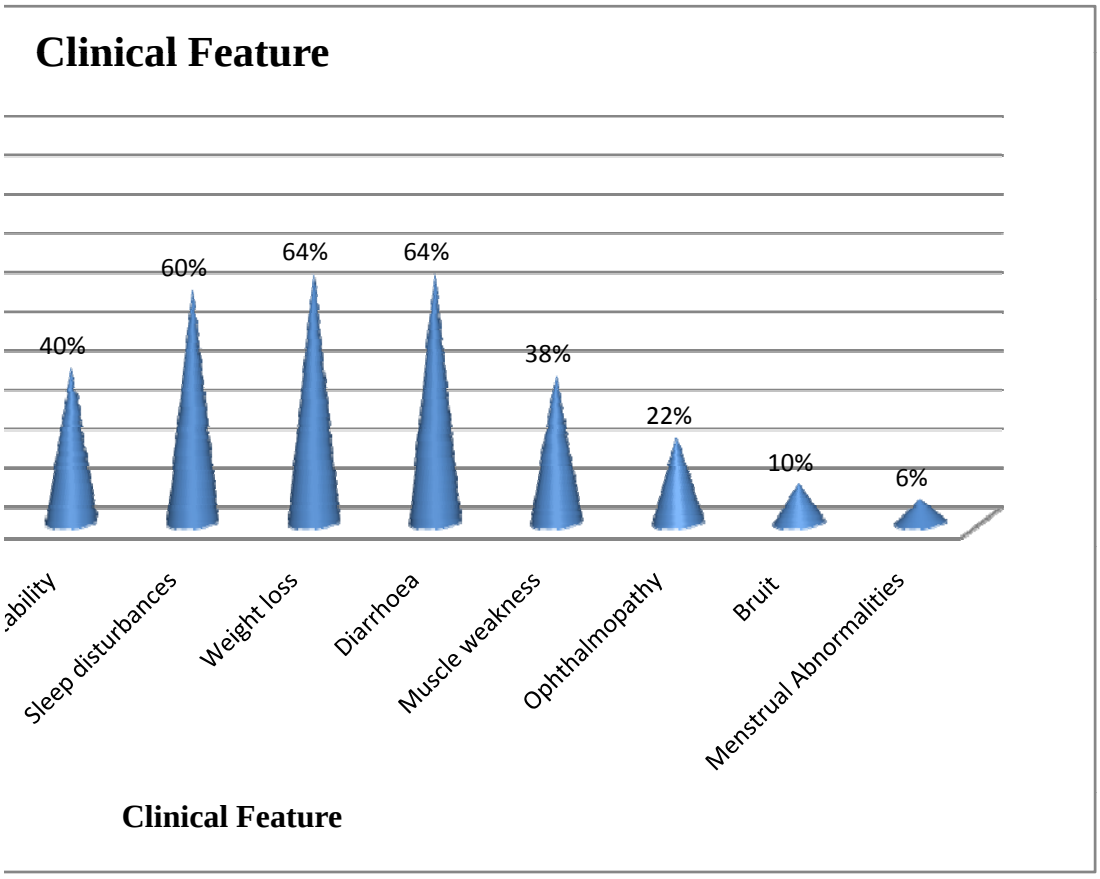


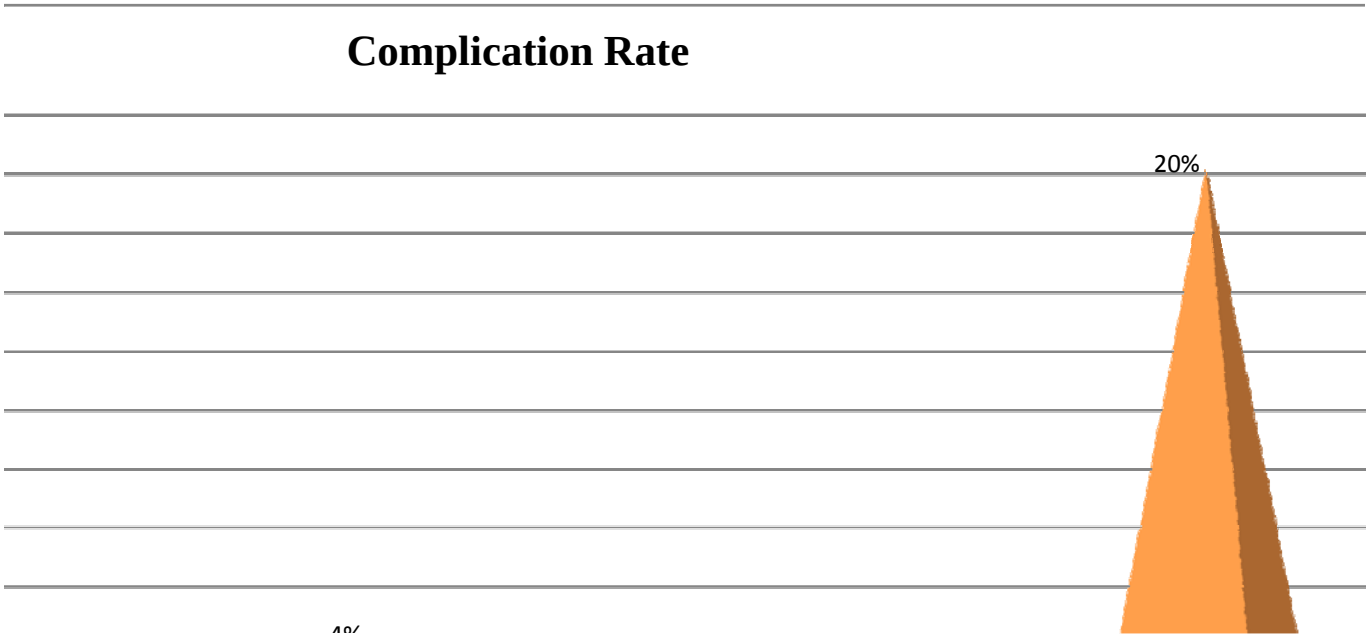


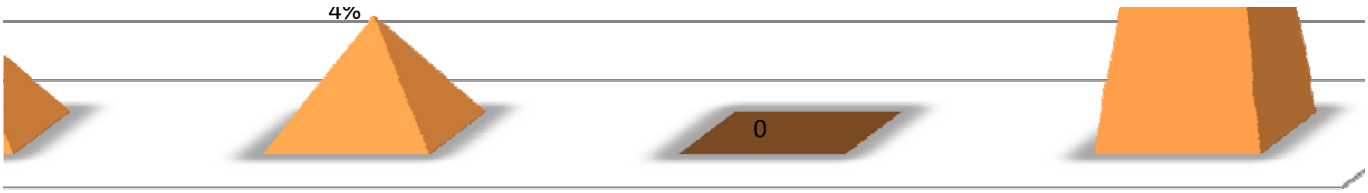


Clinical presentation


Clinical presentation
70%
30%
Primary - Diffuse toxic goitre
Secondary - Toxic MNG
Type of Goitre


Clinical Feature
40%
60%
64%
64%
38%
22%
10%
6%
ability
Sleep disturbances
Weight loss
Diarrhoea
Muscle weakness
Ophthalmopathy
Bruit
Menstrual Abnormalities
Clinical Feature

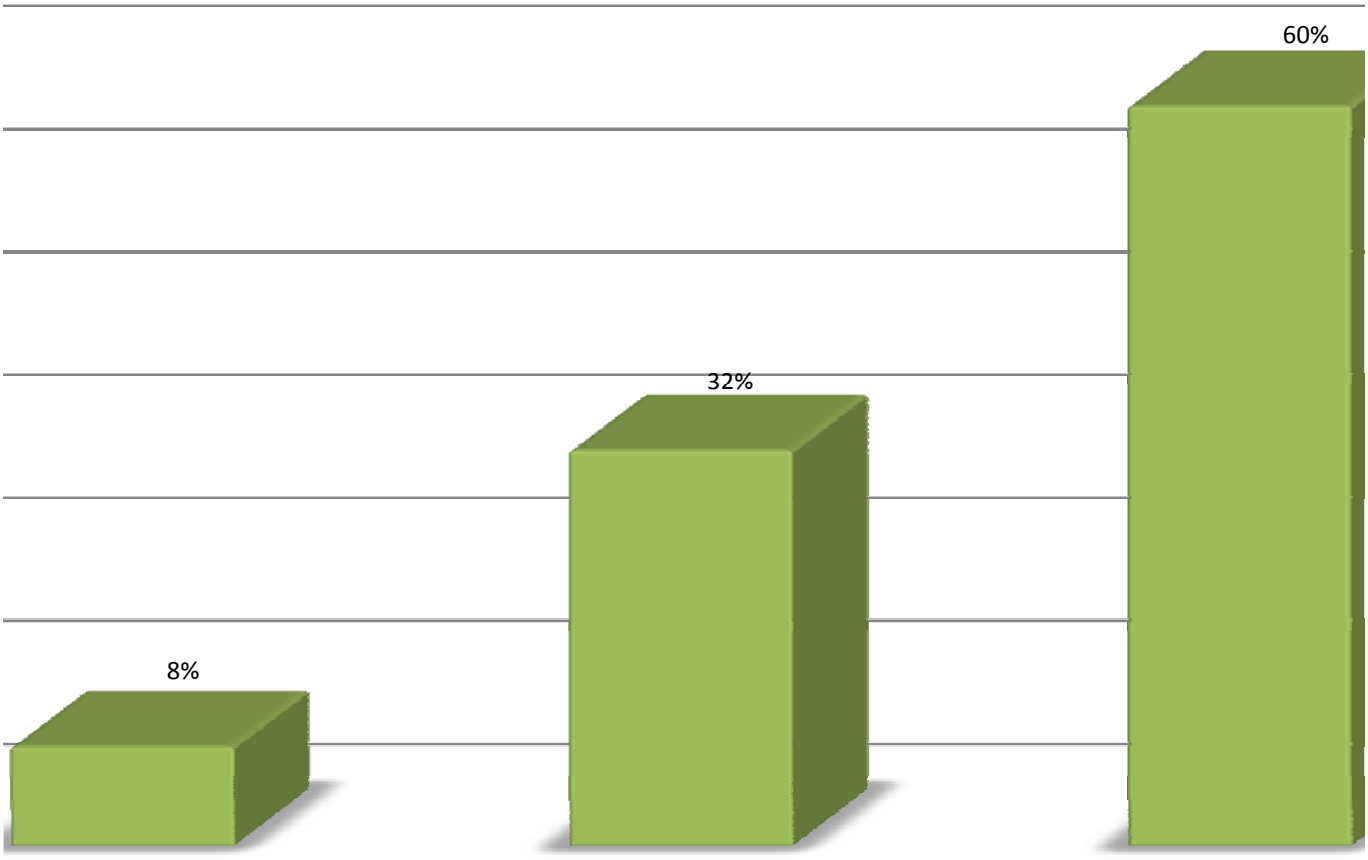

Complication Rate
20%



Hypoparathyroidism Recurrent thyrotoxicosis Hypothyroidism

Ranges of results of surgery

Grade of toxicity



<90 90-110 >110

Range